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WENDEROTH, LIND & PONACK, L.L.P.
2033 K STREET N. W.
SUITE 800
WASHINGTON, DC 20006-1021

EXAMINER	
KOSAR, AARON J	

ART UNIT	PAPER NUMBER
1651	

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Please find below and/or attached an Office communication concerning this application or proceeding.

The time period for reply, if any, is set in the attached communication.

Office Action Summary	Application No.	Applicant(s)	
	10/575,712	TAKEO ET AL.	
	Examiner	Art Unit	
	AARON J. KOSAR	1651	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE *3* MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *07 November 2007*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *18,25,26 and 28* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *18,25,26 and 28* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☒ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☒ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☒ Information Disclosure Statement(s) (PTO/SB/08) Paper No(s)/Mail Date *4/13/2006*.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application
6) ☐ Other: _____.

DETAILED ACTION

Applicant's amendment and argument filed June 2, 2008 and the supplemental responses filed August 8, 2008 and October 16, 2008 in response to the non-final rejection, are acknowledged and have been fully considered. Any rejection and/or objection not specifically addressed is herein withdrawn.

Applicant has amended the claims by amending claims 18, 25, and 26, canceling claims 1-17, 19-24, and 27, and introducing new claim 28. **Claims 18, 25, 26, and 28** are pending and have been examined on the merits to the extent of the elected invention. Election was made **without** traverse in the reply filed on November 7, 2007. The election/restriction requirement is still deemed proper and therefore made Final.

Response to Arguments

Applicant's arguments, with respect to the anticipation of the claim(s) by MORISHITA/AKINOMOTO/NAKAMURA under 35 USC § 102(b) have been fully considered and are persuasive. Therefore, the rejections have been withdrawn. However, upon further consideration, in view of the declaration and necessitated by Applicant's amendments to the claims, new grounds of rejection are made in view of TSUZUKI (**U-2**) under 35 USC § 102(b) and NAKAMURA (**AA/BA/A-2**) and SUGIMOTO (**V-2**) under 35 USC § 103(a).

Response to Amendment

The declaration under 37 CFR 1.132 filed June 2, 2008 is sufficient to overcome the rejection of claims 25 and 26 based upon 35 USC § 112, ¶1.

Claim Rejections - 35 USC § 112

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 18 and 28 are/remain rejected under 35 U.S.C. 112, first paragraph, because the specification - while being enabling for a method of treating a mammal suffering from a cerebral embolism-induced cerebrovascular hyperpermeability/edema wherein said embolism induced by injection into and occlusion of the right external carotid or pterygopalatine arteries, and said method comprising injecting into the cerebral ventricle a concentration of hepatocyte growth factor (HGF) in an amount sufficient to ameliorate symptoms associated with embolism-induced damage comprising symptoms of loss in memory, learning deficiencies, cerebrovascular hyperpermeability, or cerebral edema- does not reasonably provide enablement for the genus of all embolism-induced disorders. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and/or use the invention *commensurate in scope* with these claims.

The factors to be considered in determining whether a disclosure meets the enablement requirements of 35 U.S.C. 112, first paragraph, have been described in In re Wands, 858 F.2d 731, 8 USPQ2d 1400 (Fed. Cir., 1988). The court in Wands states, "Enablement is not precluded by the necessity for some experimentation, such as routine screening. However, experimentation needed to practice the invention must not be undue experimentation. The key word is 'undue', not 'experimentation'" (Wands, 8 USPQ2sd 1404). Clearly, enablement of a claimed invention cannot be predicated on the basis of quantity of experimentation required to make or use the invention. "Whether undue experimentation is needed is not a single, simple factual

determination, but rather is a conclusion reached by weighing many factual considerations" (Wands, 8 USPQ2d 1404). *Among these factors are: (1) the nature of the invention; (2) the breadth of the claims; (3) the state of the prior art; (4) the predictability or unpredictability of the art; (5) the relative skill of those in the art; (6) the amount of direction or guidance presented; (7) the presence or absence of working examples; and (8) the quantity of experimentation necessary.*

While all of these factors are considered, a sufficient amount for a *prima facie* case is discussed below.

The nature of the invention and the breadth of the claims:

The claims are generally drawn to the method comprising administering HGF to a any mammal suffering from embolism-induced cerebrovascular "hyperpermeability" *per se* to ameliorate any symptom associated in an undisclosed manner to any embolism-inducible condition. Claim 28 is further drawn to a mammal having cerebrovascular hyperpermeability also having cerebral edema.

The specification, however, recites a rat model wherein said rat suffers from a carotid/pterygopalatine artery occlusion and treating acute cerebrovascular leakage (FITC-albumin leakage across the blood:brain barrier (BBB)) or cerebral edema or the symptoms of declining learning and/or memory.

Thus the claims have a greater breadth than what is supported by the specification.

The state of the prior art and the predictability or unpredictability of the art:

The state of the prior art is such that HGF and the administration of HGF intraventricularly to treat/improve specific conditions (memory/learning in normal rats; stroke

remediation in afflicted mammals; etc.) is known, as taught by GHODA (**U'**: PTO-892) (e.g. Abstract)

BOJE (**U**:PTO-892) further teaches the utility of rat models and anatomy of the right common carotid artery and the interrelation with the occipital, internal carotid, pterygopalatine arteries (figures 7.19.2 and 7.19.3).

DATE (**V**:PTO-892) teaches that injection of microspheres into the internal carotid artery of rats induces a variety of manifestations by teaching that the selected animals used in a water maze test were categorized/scored and preselected such that the most severely deficient rats as measured by paucity of movement, truncal curvature, and forced circling during locomotion were selected. This constitutes a teaching that microsphere-induced embolism administered through the internal carotid artery resulted in motor skill deficiencies (mobility, posture, coordination deficiencies respectively). Date also teaches that (c. May 11,2004) that HGF "should be evaluated as a prospective agent for the therapy against ischemic brain injuries, including cerebral infarction and vascular dementia"(page 1157, concluding ¶). This constitutes a teaching that treating (vascular) dementia with HGF and as a result the effects thereof was unresolved in the art at the time of Date.

SEKIGUCHI (**W**:PTO-892) teaches that microsphere-induced cerebral ischemia may affect the ipsilateral cerebellum and medulla and the contralateral cerebellum (¶1, page 274). Sekiguchi also teaches that microsphere injection produces "widespread formation of small emboli and multiple infarct areas in the brain…including cerebral cortex, hippocampus, corpus striatum, midbrain, cerebellum, and medulla"(page 270, right column, ¶1-2). This constitutes a

teaching that microsphere-induced damage is primarily, but not isolated to, the immediate efferent arterial vicinity of the vascular injection site.

GUARCH (**X**:PTO-892) teaches that memory loss (a decline in memory function) is a "manifestation of the normal aging process" and, independent of age, may also have neither cognitive nor functional impairments. Guarch also teaches that memory loss may be asymptomatic with respect to memory loss, especially in subjective memory loss subjects where 75-90% may be asymptomatic for other deficiencies (¶3, page 353). This constitutes teaching of the unpredictability of the symptoms of memory loss and dementia.

OVERSHOT (**U-1**:PTO-892) teaches that delirium may be superimposed upon an underlying dementia and that delirium has similar symptoms as dementia (memory impairment, disorientation, and/or altered psychomotor activity) (page 491, ¶3; table 1). Overshot teaches that it is essential to assess multiple areas of cognition (page 492, last ¶) and that a full history and mental state examination should be performed although Overshot teaches that the Mini-Mental State Examination (MMSE) is commonly used. The teachings of overshot constitute a teaching (1) that dementia is complex and requires multiple indices to properly resolve versus other conditions and various forms of dementia (e.g. delirium, depression, Alzheimer's, etc.) and (2) that the tests used to diagnose and resolve dementia and/or memory are question-answer based assessments (MMSE is a 30-point/question test)(page 494, ¶2). Overshot does not teach any adapted methods for assessing memory function, learning, or dementia in non-human subjects.

ISHIKAWA (**V-1**:PTO-892) teaches that single evaluation of a drug with one learning paradigm was difficult to justify that a drug is effective for dementia (abstract). Ishikawa also teaches that "the criterion of dementia is cognitive deficits which are manifested as memory

impairment and other disturbances..", that "memory itself is not a simple phenomenon" and that "memory impaired in dementia is [declarative], which is only observed in humans" (page 39; page 43, ¶ 2). Ishikawa further teaches that learning experiments using animal models one must be careful, because the criteria for the condition, dementia, includes not only memory impairment, but also disturbances of the central nervous function, such as aphasia, apraxia, and agnosia (page 43, ¶2).

Although HGF is taught to be an angiogenic polypeptide and to have utility, the mechanism between HGF and complex conditions appears to not be fully resolved in the prior art for the genus of "embolism-induced disorders" *per se*, especially for embolism-induced disorders *per se* and including disorders such as dementia, including vascular dementia (Date, see above) or the complex conditions including memory and dementia (Overshot, see above) which may be affected by angiogenesis or some as-of-yet unidentified property of HGF upon the affected tissues/organism or for the biochemical mechanism/nexus by which HGF effects amelioration of the genus of symptoms of the myriad of embolism-induced disorders.

Since the mechanisms of inhibiting declining memory and dementia and assessing the conditions is largely unresolved as discussed above, the art is therefore highly unpredictable, except to the extent of the species of effects of record and as demonstrated in the prior art.

Furthermore, pharmaceutical therapies are unpredictable for the following reasons: (1) therapeutic compositions may be inactivated before producing an effect, i.e. such as proteolytic degradation of the peptide or protein; (2) the therapeutic composition may not reach the target area, i.e. the peptide or protein may not be able to cross the mucosa or may be adsorbed or processed by fluids, cells and tissues where the peptide or protein has no effect, (3) other

functional properties, known or unknown, may make the therapeutic composition unsuitable for *in vivo* therapeutic use, i.e. such as adverse side effects prohibitive to the use of such treatment (See page 1338, footnote 7 of Ex parte Aggarwal, 23 USPQ2d 1334 (PTO Bd. App. & Inter. 1992)).

The relative skill of those in the art:

The relative skill of those in the art is high; however with respect to the *a priori* knowledge as to predicting the effect which HGF's physiological mechanism (neovascularization inhibiting hyperpermeability to a myriad of compounds) may have upon complex genus of neurological behavioral disorders/disease states is beyond the purview of the skilled artisan except to the extent of the effects demonstrated for select species (e.g. edema, learning, memory, etc.).

The amount of direction or guidance presented and the presence or absence of working examples:

The specification has provided examples of treating a mammal, including the species of male Wistar rats, having a microsphere embolism (ME)-induced cerebrovascular accidents or ischemia, the treatment comprising injecting a solution of HGF into the right cerebral ventricle, resulting in (a) inhibition of the magnitude of loss of memory/learning and (b) inhibition of adventitious *hyperpermeability of FITC-albumin* across the BBB from the cerebrovascular milieu; however, the specification does not provide a sufficient number of working examples to be representative of inhibition of all cerebrovascular hyperpermeable events or all neurological conditions/syndromes/diseases/functions or in all instances.

The quantity of experimentation necessary:

Considering the state of the art and the high unpredictability and the lack of guidance provided in the specification, one of ordinary skill in the art would be burdened with undue experimentation to make and use the invention commensurate in scope with the claims.

It is the Examiner's position that one skilled in the art could not practice the invention commensurate in the scope of the claims without undue experimentation. It is also noted, considering the *a priori* unpredictability in the art with regard to correlating the behavioral symptoms with the physiological mechanism of HGF, that treatment to improve all embolism-induced conditions are not enabled in the manner instantly claimed.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless –
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 18, 25, 26, and 28 are rejected under 35 U.S.C. 102(b) as being anticipated by TSUZUKI (**U-2**:PTO-892: Tsuzuki N., et al. "Hepatocyte Growth Factor Reduces the Infarct Volume After Transient Focal Cerebral Ischemia in Rats", Neurological Research, 23(4), June 2001 , pages 417-424.)

The claims are as of record and as above. The claims are drawn in general to a method comprising a step of injecting hepatocyte growth factor (HGF) into a mammal suffering from an embolytic-induced cerebrovascular hyperpermeability to remediate a symptom having an association with a disorder.

TSUZUKI (U-2) anticipates the claims by teaching intraventricularly administering/ injecting mammals (e.g. rats) suffering from transient focal cerebral ischemia with HGF. Tsuzuki further teaches that said ischemia comprises arterial occlusion/embolism in the right middle

cerebral artery and the bilateral common carotid arteries wherein said ischemia affects neuronal cells of the CNS. HGF prevented neuronal death and reduced the infarct volume (edema) in a dose-dependent manner. Tsuzuki further teaches that the result of HGF injection provides "an ability to prevent apoptotic neuronal cell death while also possessing an angiogenic effect in the central nervous system which was affected with transient focal cerebral ischemia"(Abstract; page 418, ¶1).

To the extent that the method of Tsuzuki may be silent with respect to a particular symptom or improvement thereof, because the affected organism (mammal, including rats), mode of treatment and composition (administering HGF intraventricularly), and location of embolism/occlusion (carotid artery) taught by Tsuzuki are identical to the instant invention to the extent claimed, the method of Tsuzuki would thus provide the dose-dependent response inherent to the chemical functioning of HGF, the dose-dependent reduction of infarct volume, and the prevention of neuronal cell-death.

Please note, as discussed above, while discovery of the biological mechanism behind the administration of a known bioactive compound is clearly publishable in a peer-review journal, the criteria for patenting claims are distinct from publication criteria. For example, if the active step of the method is the same and the subject is the same, then the claimed method can be anticipated or made obvious by the prior art, even if the prior art does not recognize or appreciate this mechanism as long as the compound and/or dose administered, mode of administration, subject, etc. are the same as in the method disclosed in the prior art. In the absence of objective evidence to the contrary or evidence as to the criticality of a specific dose, the administration of the HGF in the manner disclosed in the prior art would be sufficient to ameliorate a symptom to

the extent claimed, especially in the absence of evidence as to the criticality of a particular concentration/range and in the absence of objective evidence as to how the symptom per se is ameliorated by some undisclosed effect/mechanism which would preclude the composition of the prior art from functioning in the instantly claimed manner.

If this were not so, one patent might issue with a one step claim of administering the a compound to a subject in order to empirically treat a specific disease which is result of a contemporaneously unknown, disordered mechanism or pathway; and, then upon later discovery of the mechanism of the disorder, additional patents could issue with one-step claims directed to the administration of the same compound to the same subject in order to modulate the specifically disordered mechanism or pathway. This would lead to multiple patents with essentially the same invention being patented, merely being couched in different words.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names *joint inventors*. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 18, 25, 26, and 28 are rejected under 35 U.S.C. 103(a) as being obvious over NAKAMURA (**AA** or **BA** or **A-2**:PTO-892: US 2003/0060403 A1) in view of SUGIMOTO (**V-2**:PTO-892: Sugimoto, N., et al. "Vagotomy Does Not Affect Thermal Responsiveness to

Intrabrain Prostaglandin E_2 and Cholecystokinin Octapeptide", Brain Research, 1999, 844(1-2), pages 157-163.).

The claims are as of record and as above.

NAKAMURA (AA) teaches a method of administering HGF and the intended uses of treating dementia and cerebral stroke/infarction. Nakamura (AA) is considered redundant to the teachings of Nakamura (A-2) which is evidence of and considered redundant to the original disclosure and Nakamura (BA).

SUGIMOTO (V-2) teaches that intrabrain administration includes intracerebroventricular (i.c.v.) injecting (e.g. §2.2-2.3).

To the extent Nakamura may be silent with respect to all of the possible effects of the administration of HGF, since the chemical properties of HGF are intrinsic to the compound, administration of the compound to a subject would be expected to have an intrinsic effect/response in similar method conditions (e.g. "treating a mammal with HGF", (BA) page 15, ¶ 1; claims 5, 7, 8, 10, 12, and 13).

To the extent the prior art may be silent with respect to a recitation of the intended use of the claimed invention (e.g. decline in learning, memory, etc.), the recitation/intended use must result in a structural difference between the claimed invention and the prior art in order to patentably distinguish the claimed invention from the prior art. If the prior art structure is capable of performing the intended use, then it meets the claim.

To the extent that Nakamura may be silent with respect to i.c.v. administration, it would have been obvious to have provided HGF intracerebroventricularly, because i.c.v. injection and injection of HGF stroke- and infarct-affected mammals are known. One would have been

motivated to have used i.c.v. injection because Nakamura teaches HGF administration by a variety of routes including intrabrain (column 6, ¶ 3) including for the treatment of cerebral conditions (e.g. cerebral stroke/infarction, column 6, ¶4) and because Sugimoto teaches that intrabrain administration embraces i.c.v. injection. Furthermore, one would recognize that other modes of injection (e.g. intravenous and intraarterial) in a cerebral stroke or cerebral infarction would comprise a compromised blood-brain barrier (anastamosis of the vascular and ventricular systems at the point of stroke/infarct trauma) and thus injecting into one system would be expected to inject the material into all compromised compartments/systems, especially in the absence of objective evidence to the contrary. One would have had a reasonable expectation of success in providing HGF by i.c.v. injection, because success merely requires substituting known delivery routes for the known and predictable result of drug delivery, and especially in the absence of objective evidence to the contrary or criticality of some undisclosed feature.

A reference is good not only for what it teaches by direct anticipation but also for what one of ordinary skill in the art might reasonably infer from the teachings. (*In re Opprecht* 12 USPQ 2d 1235, 1236 (Fed Cir. 1989); *In re Bode* 193 USPQ 12 (CCPA) 1976). In light of the forgoing discussion, the Examiner concludes that the subject matter defined by the instant claims would have been obvious within the meaning of 35 USC 103(a).

From the teachings of the references, it is apparent that one of ordinary skill in the art would have had a reasonable expectation of success in producing the claimed invention. Therefore, the invention as a whole was *prima facie* obvious to one of ordinary skill in the art at the time the invention was made, as evidenced by the references, especially in the absence of evidence to the contrary. Also please note, Nakamura (AA/BA/A-2), relied upon herein, issued

as US PAT. 6,699,837 (BA) prior to December 10, 2004 (pre-CREATE Act) and was filed (see A-2) prior to November 29, 1999 (pre-AIPA).

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire THREE MONTHS from the mailing date of this action. In the event a first reply is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to AARON J. KOSAR whose telephone number is (571)270-3054. The examiner can normally be reached on Monday-Thursday, 7:30AM-5:00PM, ALT. Friday,EST.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Mike Wityshyn can be reached on (571) 272-0926. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free). If you would like assistance from a USPTO Customer Service Representative or access to the automated information system, call 800-786-9199 (IN USA OR CANADA) or 571-272-1000.

/Aaron Kosar/
Examiner, Art Unit 1651

/Sandra Saucier/
Primary Examiner, Art Unit 1651